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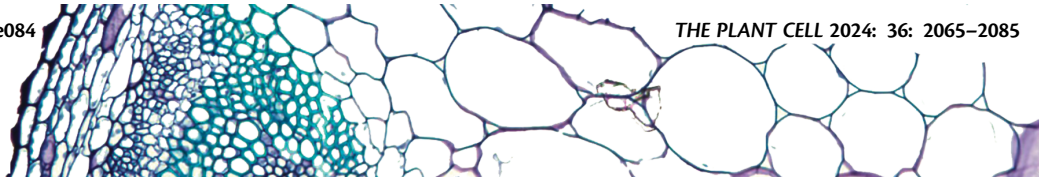

Review

What is going on inside of phytochrome B photobodies?

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Abstract

Plants exhibit an enormous phenotypic plasticity to adjust to changing environmental conditions. For this purpose, they have evolved mechanisms to detect and measure biotic and abiotic factors in their surroundings. Phytochrome B exhibits a dual function, since it serves as a photoreceptor for red and far-red light as well as a thermosensor. In 1999, it was first reported that phytochromes not only translocate into the nucleus but also form subnuclear foci upon irradiation by red light. It took more than 10 years until these phytochrome speckles received their name; these foci were coined photobodies to describe unique phytochrome-containing subnuclear domains that are regulated by light. Since their initial discovery, there has been much speculation about the significance and function of photobodies. Their presumed roles range from pure experimental artifacts to waste deposits or signaling hubs. In this review, we summarize the newest findings about the meaning of phyB photobodies for light and temperature signaling. Recent studies have established that phyB photobodies are formed by liquid-liquid phase separation via multivalent interactions and that they provide diverse functions as biochemical hotspots to regulate gene expression on multiple levels.

Introduction to phytochrome B

Plant genomes provide the foundation for a striking phenotypic plasticity in response to changes in their environment. While extreme light and temperature conditions can indicate a stressful environment for plants, daily and annual changes of both factors provide important information about the current season and the time of day. Light is one of the most essential environmental factors for plants since it not only serves as an environmental cue but also as their energy source. Therefore, plants have evolved sophisticated systems of photoreceptors to monitor their light environment, which allows them to optimize their growth and development accordingly.

Phytochromes are a family of photoreceptors that can be found in bacteria, fungi, and the entire green lineage from algae to flowering plants. *Arabidopsis thaliana*'s genome encodes for 5 phytochromes that act as red (R) and far-red (FR) light receptors to sense light intensity and quality. Mutant analyses demonstrated that phytochrome B (phyB) is the major phytochrome in *Arabidopsis* and that it is involved in plant development processes throughout the entire plant life cycle, from germination and seedling de-etiolation to floral induction and seed yield (Franklin and Quail 2010; Ulijasz and Vierstra 2011; Legris *et al*. 2019).

The PHYB apoprotein has a mass of approximately 125 kDa and can be divided into 2 larger domains (or modules) that are linked through a flexible hinge region: the N-terminal photosensory (or signal input) domain and the C-terminal output domain that is necessary for homodimerization or heterodimerization with other phytochromes. These 2 domains can be further divided into several subdomains (Fig. 1). The photosensory domain consists of 4 subdomains: N-terminal extension, PAS (Period/Arnt/Single-Minded) domain, GAF (cGMP phosphodiesterase/adenylyl cyclase/FhlA) domain, and PHY (Phy-specific) domain. The output module is composed of a PAS-related domain and a HKRD (histidine kinase-related domain). The latter subdomain is a feature that points at the bacterial origins of phytochromes as photo-regulated histidine kinases

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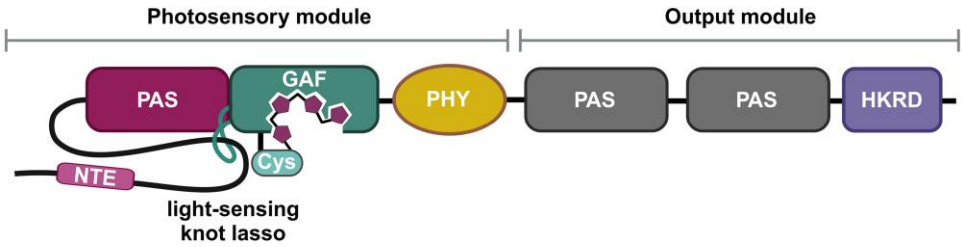

Figure 1. Domain structure of *Arabidopsis* phyB. The photosensory domain consists of a PAS domain, a GAF domain, and a PHY domain. The output module is composed of a PAS-related domain and a HKRD. The C-terminal output module features properties for nuclear localization, dimerization and photobody formation, while the N-terminal photosensory module binds phytochromobilin via a conserved cysteine residue (Cys) and possesses the N-terminal extension (NTE) that is required for photobody formation of the full-length phyB protein. The depicted domain structure is based on (Burgie and Vierstra 2014).

(Rockwell *et al.* 2006; Nagatani 2010; Burgie and Vierstra 2014).

Bacterial phytochromes dimerize in linear and symmetric head-to-head fashion (Li *et al.* 2010). In contrast, phyB dimers exhibit an asymmetric architecture: in the dimer, the head-to-head association is only retained for the C-terminal histidine-related domains, whereas the N-terminal photosensory modules are arranged head-to-tail (Li *et al.* 2022b). Interestingly, this likely implies that during evolution, conserved interacting protein surfaces within the photosensory modules must have been abandoned and new ones must have been established to give rise to the PHYB protein clade of phytochromes.

After translation, a chromophore (phytochromobilin) binds covalently to the GAF domain of the photosensory module to form the phyB holoprotein. Absorption of R or FR light triggers the isomerization of the phytochromobilin that transmits into conformational changes of the phyB protein. This allows phyB to reversibly transition between 2 relatively stable conformers: R light induces the biologically active Pfr form, while FR light triggers the transition of Pfr into the Pr conformation, which represents inactive phyB (Klose *et al.* 2015). The Pfr form is temporarily stable even after the transition to darkness during the onset of night. Here, phyB slowly switches back into its inactive Pr form. This process is called dark reversion or thermal reversion, since it is accelerated by elevated temperatures (Klose *et al.* 2020). Therefore, phyB exhibits a dual function: not only does it act as R/FR light receptor but also as a thermosensor during the night (Jung *et al.* 2016) and the day (Legris *et al.* 2016; Qiu *et al.* 2019). Additionally, phyB is also able to detect other plants that are potential competitors for sunlight. Photosynthetically active tissues absorb more R than FR light, since the R light is utilized for photosynthesis. In contrast, most of the FR light transmits through or is reflected from leaves of neighboring plants. Therefore, a plant that is surrounded by potential competitors perceives light with a low R to FR light ratio (low R:FR). This reduces phyB activity and hence allows the detection of neighboring vegetation before being exposed to the vegetation's shade. In consequence, shade-avoiding plants like *Arabidopsis* induce an escape strategy called shade avoidance response that promotes stem and petiole elongation—to outgrow potential competitors—and that induce early flowering (Casal 2012; Ballaré and Pierik 2017). The negative effect on phyB activity is intensified if the plant is exposed to true canopy shade that not only increases the amount of perceived FR light but also decreases the photosynthetically active radiation (PAR), including R light. In consequence, the reduction of the R to FR light ratio, the decrease of phyB activity, and the extent of the shade responses are intensified in true shade conditions (Fiorucci and Fankhauser 2017; Hernando *et al.* 2021; Martinez-Garcia and Rodriguez-Concepcion 2023).

What are phyB photobodies?

In etiolated seedlings, phyB is inactive and resides within the cytosol. However, the exposure to R light converts phyB into its active Pfr form, which triggers a rise of the cytoplasmic Ca^{2+} concentration (Zhao *et al.* 2023). This Ca^{2+} increase activates 2 calcium-dependent kinases that bind and phosphorylate phyB. Their activity induces the translocation of phyB into the nucleus, where the photoreceptor initiates the physiological responses to R light (Zhao *et al.* 2023). Under dim R light, phyB is evenly distributed within the nucleoplasm. However, with increasing light intensity, phyB starts to form nuclear speckles or nuclear bodies (Chen *et al.* 2003). These nuclear speckles formed by phytochromes were first reported in 1999 by Akira Nagatani, Ferenc Nagy, Eberhard Schäfer, and colleagues (Kircher *et al.* 1999; Yamaguchi *et al.* 1999). While other light receptors are also able to form nuclear speckles, the term photobodies was originally coined by Joanne Chory in 2011 to specifically describe phytochrome containing nuclear bodies (Chen and Chory 2011). Photobodies formed by phyB are dynamic and increase in size with rising light intensity (Fig. 2). Weaker red light forms many small nuclear speckles, an intermediate R light intensity leads to the production of many small and a few larger photobodies, while strong, saturating R light confines phyB into a handful of large bodies. In contrast, lowering the R light intensity or exposure to low R:FR leads to the opposite behavior. Both promote the

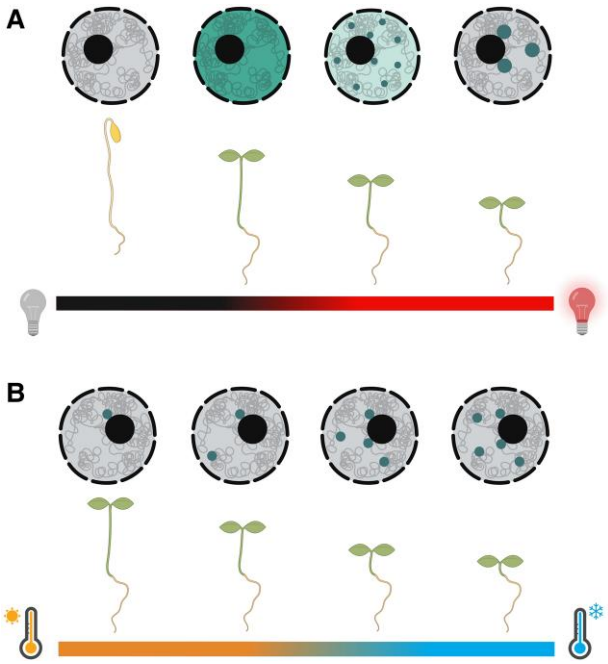

Figure 2. Schematic illustration of photobody behavior under different red light and temperature conditions. **A)** Under very dim red light, phyB translocates into the nucleus where it is evenly distributed. An increasing fluence rate leads to small photobodies that fuse and form bigger nuclear bodies with rising light intensities. **B)** In constant red light, increasing temperatures cause the disassembly of individual photobodies without disintegrating into small nuclear foci. Thermolabile photobodies are only present in lower temperatures, while the most thermostable photobody is located in the proximity of the nucleolus.

redistribution of phyB from large photobodies into smaller speckles or even into the nucleoplasm (Chen *et al.* 2003; Trupkin *et al.* 2014; Van Buskirk *et al.* 2014; Willige *et al.* 2021). Additionally, photobodies are also temporally regulated. Within minutes, light exposure of etiolated seedlings leads to the formation of early photobodies that are transient and small. In contrast, prolonged light exposure leads to the formation of large and stable photobodies (Bauer *et al.* 2004). Altogether, smaller photobodies seem to be an intermediate step from phyB's redistribution from the nucleoplasm into large bodies or vice versa. Similarly, the size of photobodies has been reported to decrease in elevated temperatures in diurnal white light conditions (Legris *et al.* 2016; Murcia *et al.* 2021). However, in constant red light, inactivation of phyB by elevated temperatures does not seem to mimic the photobody dynamics in response to changes in light intensity or quality. Instead, increasing temperatures lead to the complete disassembly of individual photobodies without the transition to small speckles (Hahm *et al.* 2020). Here, each photobody within a cell seems to have an unique thermal sensitivity. This demonstrates that photobodies within a cell are not equal, which is supported by the observation that a photobody located in proximity to the nucleolus is most stable (Fig. 2B). Interestingly, when phyB is constitutively active by overexpressing the phyB Y276H mutant (YHB), cotyledon photobodies are shown to be temperature insensitive, indicating that photobody sensitivity is linked to phyB's reversion into its inactive Pr state (Hahm *et al.* 2020). This observation contrasts with another study that shows temperature sensitivity of photobodies formed by YHB in hypocotyl or HEK293T cells. This would indicate that the temperature sensitivity of photobodies is additionally based on a thermal reversion–independent mechanism (Chen *et al.* 2022). Future studies will show if these contradicting results are the consequence of different *PHYB* expression levels or analyzed cell types. However, both studies agree that the successive disassembly of a cell's photobodies could represent a sensor to assess increasing ambient temperatures (Hahm *et al.* 2020; Chen *et al.* 2022). Besides light and temperature, photobody stability is also regulated by other external and internal factors such as salt stress or jasmonate signaling (Liu *et al.* 2023; Peng *et al.* 2023). This indicates that photobodies have the ability to integrate multiple signaling pathways.

How are these dynamics in photobody assembly and disintegration achieved? Two phyB (sub)domains are required for the formation of nuclear bodies. It has been reported that the C-terminal output module alone is sufficient to dimerize, translocate into the nucleus, and form photobodies even in the dark (Nagy *et al.* 2000; Matsushita *et al.* 2003). This indicates that the N-terminal photosensory domain prevents the translocation and speckle formation of phyB's Pr form. However, the full-length protein in its Pfr form requires the N-terminal extension to assemble into nuclear foci (Chen *et al.* 2005). This implies a positive regulatory role of the photosensory domain in speckle formation as well. The N-terminal extension is important to stabilize the Pfr form of phyB by slowing down the dark/thermal reversion, and it harbors an intrinsically disordered region (Burgie *et al.* 2021). In general, intrinsically disordered regions are often a requirement for biomolecular condensation, since they allow multivalent interactions to form large protein complexes (Emenecker *et al.* 2020; Borcherds *et al.* 2021). Similarly, photobody assembly requires the formation of higher order phyB aggregates through interactions of multiple phyB dimers through their N-terminal extensions (Chen *et al.* 2022).

As stated above, these photobodies are dynamic, since fluorescence recovery after photobleaching experiments indicated that the phyB molecules move from photobody to photobody through the nucleoplasmic phyB pool (Rausenberger *et al.* 2010). Furthermore, photobodies are spherical in shape and can move and coalesce (Chen *et al.* 2022). Therefore, it has been suggested that photobodies are formed through the formation of a dense phyB phase (photobodies) within a less dense phyB phase (nucleoplasm) (Fonin *et al.* 2021; Chen *et al.* 2022). This process is called phase separation and allows the dynamic assembly of membraneless organelles within the nucleus (Emenecker *et al.* 2020).

So far, there are 2 estimates of the number of phyB dimers per photobody. Due to the chosen experimental approach or due to photobody dynamics, both estimates provide quite different results: 80 to 150 phyB dimers per 0.4- to 0.8-μm^3 photobody and 1,500 dimers per 0.2-μm photobody (Chen *et al.* 2022; Kim *et al.* 2023a). Certainly, photobodies consist not only of phyB but of a complex mixture of diverse proteins. A recent mass spectrometry study that used photobodies isolated by fluorescence-activated particle sorting to gain insights into their composition revealed that photobody clients can be classified into 2 groups: proteins that bind phyB directly and proteins that require the presence of the first group to be targeted to photobodies (Kim *et al.* 2023a). However, the individual photobodies of a single cell are not all the same and do not carry the same protein composition, which is supported by phyB colocalization studies. For example, the blue light receptor cry2 also forms nuclear speckles, but only a subset of these speckles within a nucleus colocalizes with phyB photobodies (Más *et al.* 2000). This indicates that different photobodies not only have differing thermal sensitivities but are also functional divers.

But what is the significance and function of photobodies? In light conditions where phyB is unable to form photobodies, only very weak R light responses are executed. This suggests that photobodies are not essential for phyB activity, but that they represent a mechanism to potentiate phyB signaling (Chen *et al.* 2003). In addition, several mutations within PHYB or of structural phyB photobody components demonstrate a causal relationship between photobody formation and phyB activity (Qiu *et al.* 2017; Huang *et al.* 2019).

In general, research on photobodies collectively indicates that photobodies serve 2 main functions. First, they stabilize phyB's Pfr form and extend the time of phyB activity. Second, they regulate biochemical reactions by concentrating or sequestering enzymes, substrates, and other factors that give these reactions specificity. By this means, phyB inactivates repressors of photomorphogenesis but promotes the activity of positive signaling components. In consequence, phyB regulates different steps of gene expression, including chromatin remodeling, transcription, and pre-mRNA splicing via photobodies. The newest findings about the role of phyB photobodies for light and temperature signaling are summarized below.

PHYTOCHROME-INTERACTING FACTORs are transcription factors regulated by photobodies

Diverse transcription factors and other transcriptional regulators that positively regulate light signaling are found in nuclear bodies—for example, HY5 (ELONGATED HYPOCOTYL 5), HFR1 (LONG HYPOCOTYL IN FAR-RED 1), ELF3 (EARLY FLOWERING 3), and several B-BOX DOMAIN PROTEINs, including BBX4, 21, and 22 (Ang *et al.* 1998; Jang *et al.* 2005; Datta *et al.* 2006, 2007, 2008; Yu *et al.* 2008). However, the transcription factor family that is best understood in phyB signaling and photobody function is the PIF (PHYTOCHROME-INTERACTING FACTOR) family (Duek and Fankhauser 2005; Leivar and Quail 2011), consisting of a group of 8 basic-helix-loop helix transcription factors. PIFs interact with photoactivated phyB via their Active-PHYB Binding (APB) motif (Khanna *et al.* 2004). In contrast to PIL1 and PIL2 (PIF3-Like1 and 2, also called PIF2 and 6) (Roig-Villanova *et al.* 2006; Penfield *et al.* 2010; Luo *et al.* 2014), PIF1, 3, 4, 5, 7, and 8 function as antagonists of phytochrome signaling (Huq and Quail 2002; Kim *et al.* 2003; Bauer *et al.* 2004; Fujimori *et al.* 2004; Huq *et al.* 2004; Oh *et al.* 2004, 2020; Li *et al.* 2012). PIFs suppress photomorphogenesis by inhibiting chloroplast biogenesis, stimulating hypocotyl growth, and promoting apical hook formation and maintenance (Leivar *et al.* 2008b; Shin *et al.* 2009; Stephenson *et al.* 2009). Additionally, PIFs also regulate diverse growth and developmental processes throughout a plant's life cycle, especially in response to environmental cues (Leivar and Quail 2011; Balcerowicz 2020; Sharma *et al.* 2023). PIFs not only mediate the light and temperature information given by light receptors but also integrate these environmental cues and intrinsic hormonal pathways. Connections to most plant hormonal pathways have been identified, including gibberellin (de Lucas *et al.* 2008; Feng *et al.* 2008), brassinosteroid (Oh *et al.* 2012; Bernardo-García *et al.* 2014), auxin (Franklin *et al.* 2011; Sun *et al.* 2012), ethylene (Khanna *et al.* 2007; Liu *et al.* 2017b), cytokinin (Richter *et al.* 2010; Aizezi *et al.* 2022), abscisic acid (Kim *et al.* 2016; Qi *et al.* 2020), and jasmonate (Yang *et al.* 2012; Fernández-Milmanda *et al.* 2020). Here, PIFs either regulate the transcription of hormone biosynthesis, transport and signaling genes or physically interact with hormone signaling components. Good examples are the various interactions with the auxin pathway. For instance, PIFs induce the expression of *YUCCA* genes to promote auxin biosynthesis (Li *et al.* 2012; Sun *et al.* 2012) and the transcription of auxin transport activating AGC kinases (Willige *et al.* 2012; Park *et al.* 2019). Furthermore, PIFs interact with ARF (AUXIN RESPONSE FACTOR) and AUX/IAA transcriptional regulators (Oh *et al.* 2014; Xi *et al.* 2021).

PIFs mainly act in concert with each other, showing at least partial redundancy for the different physiological responses they execute. An example of this redundancy can be seen in hypocotyl growth in response to warm temperatures and low R:FR. These growth responses are mainly stimulated by PIF4, 5, and 7, but PIF4 plays the major role in the context of thermomorphogenesis, while PIF7 is the master regulator in low R:FR and in conditions in which warm temperatures and low R:FR are perceived simultaneously (Burko *et al.* 2022).

Like the classification of phytochromes as photolabile (phyA-type) and photostable (phyB-type) (Clough and Vierstra 1997), PIFs also fall into 2 separate groups. While PIF1, 3, 4, 5, and 8 are destabilized by R light and phyB activity (Bauer *et al.* 2004; Park *et al.* 2004; Shen *et al.* 2005, 2007;

Lorrain *et al.* 2008; Oh *et al.* 2020), PIF7 protein levels are stable in response to R light and are not negatively affected by phyB (Leivar *et al.* 2008a; Willige *et al.* 2021). Independently of their photostability, photoactivated phyB recruits PIFs to photobodies (Fig. 3). Here, binding by phyB leads to inactivation and phosphorylation of PIFs and subsequently polyubiquitination and, eventually, degradation of red light labile PIFs (Bauer *et al.* 2004; Al-Sady *et al.* 2006; Shen *et al.* 2008; Ni *et al.* 2013, 2014, 2017). Furthermore, PIFs also seem to play a role in phyB photobody formation. This is supported by the absence of early photobodies in *pif3* mutants during de-etiolation in R light (Bauer *et al.* 2004). In addition, both phyB and PIF7 can independently form nucleolus localized speckles when tethered to nucleoli. Interestingly, when only PIF7 is tethered to nucleoli, it recruits phyB into its speckles, which indicates that PIF7 helps photobody initiation/stabilization (Liu *et al.* 2014). This is supported by a recent study, where phyB formed faster and larger photobodies in a *PIF7* overexpressor line after the transition from low R:FR to white light (Xie *et al.* 2023).

The founding member of the PIF family, PIF3, was originally identified in a yeast 2-hybrid screen using the C-terminal output module of phyB as bait protein (Ni *et al.* 1998). Nevertheless, PIFs do not only interact with phyB's output module but also with the light-sensing knot lasso located in the photosensory domain (Ni *et al.* 1999; Kikis *et al.* 2009). It was proposed that only the latter interaction is crucial for phyB signaling and PIF inactivation and degradation; deleting the output domain and forcing the remaining photosensory module to dimerize in the nucleus generates a protein that can trigger various phyB responses without being able to form photobodies (Matsushita *et al.* 2003; Oka *et al.* 2008). This suggested that the output domain and photobodies are dispensable for phyB signaling. Nevertheless, subsequent studies demonstrated that PIF interaction with both phyB modules is necessary to execute proper phyB signaling. Using the dimerizing photosensory domain revealed that this N-terminal module sequesters PIF3 and prevents its DNA binding without inducing PIF phosphorylation and degradation (Park *et al.* 2012). Additionally, this interaction with phyB's N-terminal photosensory module suppresses PIF3's transcriptional activity, which is mediated by a single, evolutionary conserved transactivation domain (Yoo *et al.* 2021). However, phyB without its C-terminal output module does not properly inhibit hypocotyl elongation in growth conditions with light/dark cycles. Furthermore, a phyB C-terminal mutation that prevents phyB dimerization and photobody formation revealed that it is the output module that is responsible for the induction of PIF phosphorylation and degradation (Qiu *et al.* 2017; Paik *et al.* 2019). The N-terminal sequestration function seems to mediate phyB's fast response after photoactivation, while the C-terminal interaction initiates a more extended response that leads to a more profound outcome: the degradation of PIF transcription factors (Park *et al.* 2018). The sole exception seems to be PIF7 that does not accumulate in phyB mutants and that is not degraded by R light (Leivar *et al.* 2008a; Willige *et al.* 2021). Therefore, PIF7 seems to form a stable PIF pool that allows quick responses after phyB inactivation without the necessity of de novo PIF translation. This could explain why PIF7 can bind to its DNA target sites within 5 minutes of low R:FR light exposure (Willige *et al.* 2021). However, PIF7 is sequestered into photobodies and is phosphorylated by phyB activity, indicating that both phyB's N- and C-terminal modules are also relevant for PIF7 inactivation. Hence, photobodies serve to inactivate all PIFs by posttranslational modifications and for this purpose diverse enzymes are recruited into photobodies.

Recruitment of PIF modifying kinases and phosphatases by photobodies

The current model proposes that PIFs bound by phyB are first phosphorylated and subsequently polyubiquitinated to induce their degradation by the 26S proteasome (Al-Sady *et al.* 2006; Lorrain *et al.* 2008; Ni *et al.* 2013). This raises the possibility that phyB itself phosphorylates the PIF proteins, since bacterial (and fungal) phytochromes bear histidine kinase activity. Interestingly, phyB carries this heritage in its HKRD at its C terminus, which is necessary for PIF phosphorylation (Qiu *et al.* 2017). However, not all amino acid residues that are crucial for the catalytic activity of histidine kinases are present in phytochrome HKRDs (Boylan and Quail 1996), and too tight packing of phyB's ATP-binding pocket could prevent a potential kinase activity (Li *et al.* 2022b). In addition to the HKRD, also the hinge region that links both phytochrome modules, the PAS-related domain as well as the photosensory module were proposed to bind ATP to confer serine/threonine kinase activity of plant phytochromes (Wong and Lagarias 1989; Yeh and Lagarias 1998; Shin *et al.* 2016). Nevertheless, the significance of a potential phyB kinase activity for its signaling processes has been debated for decades.

Several kinases have been reported to phosphorylate PIFs. The MLK/PPK (MUT9P-LIKE-KINASE/PHOTOREGULATORY PROTEIN KINASE) family form a small group of nuclear localized Casein Kinases 1-like proteins that regulate hypocotyl growth and flowering time. The kinases were co-purified as an interactor of PIF3 and phyB in a R light-induced manner. Furthermore, in the dark, MLK4/PPK1 is evenly distributed in the nucleoplasm but concentrates in nuclear speckles after a R light exposure (Ni *et al.* 2017). However, the recruitment of MLK1/PPK2, MLK2/PPK3, and MLK4/PPK1 into photobodies requires the presence of PIF3. The transcription factor serves as a primary client that is required to relocate MLKs/PPKs into photobodies (Kim *et al.* 2023a).

Loss of MLK/PPK function attenuates the light-dependent phosphorylation and degradation of PIF3. Additionally, in vitro assays demonstrate that MLK4/PPK1 phosphorylates PIF3 at amino acid residues that were previously identified as light-inducible phospho-sites. Altogether, these results

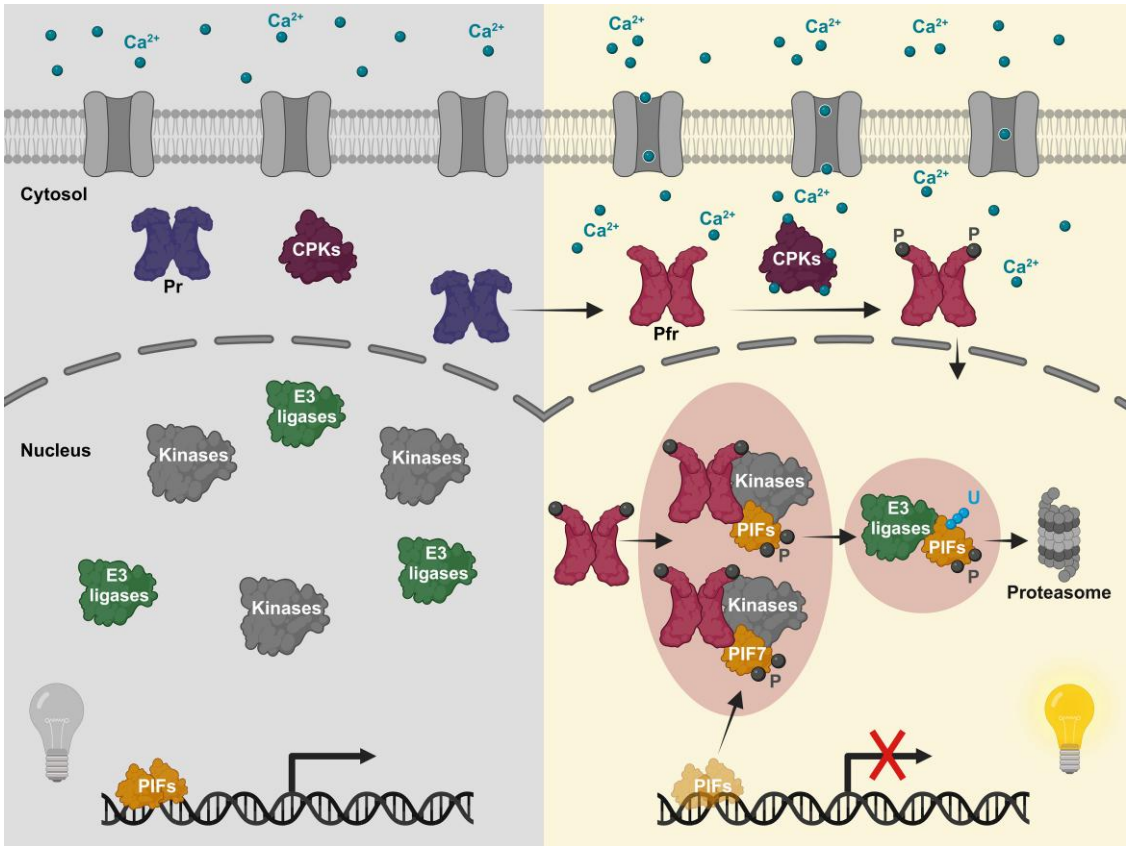

Figure 3. In the dark, phyB is located in the cytoplasm in its inactive Pr conformation. Hence, PIFs are abundant and active to regulate their target genes. Upon light activation and conformational switch to its Pfr form, phyB activates calcium channels by an unknown mechanism. Hence, an increased intracellular Ca^{2+} concentration leads to activation of CPKs (CALCIUM-DEPENDENT PROTEIN KINASEs) that phosphorylate the light receptor and induce its nuclear translocation where it inactivates PIFs. Subsequently, PIFs are phosphorylated and ubiquitinated to determine their degradation via the 26S proteasome. PIF phosphorylation, ubiquitination, and degradation are mediated by the output module, but it is unclear if these processes are all happening within the same photobody. PIF7 is only phosphorylated and inactivated by phyB activity but not targeted for degradation. U denotes ubiquitination, while P indicates phosphorylation events.

make the MLK/PPK family a good candidate for a phytochrome-activated kinase that induces PIF degradation (Ni *et al.* 2017). However, surprisingly, higher order *mlk*/*ppk* mutants show a reduced hypocotyl growth in contrast to the expected R light hyposensitivity that would go along with PIF stabilization (Huang *et al.* 2016a; Ni *et al.* 2017). It was proposed that the underlying cause for the repressed growth are the higher phyB protein levels in *mlk*/*ppk* mutants. Alternatively, MLKs/*PPK*s have pleiotropic functions (Wang *et al.* 2015, 2021; Liu *et al.* 2017a; Wirthmueller *et al.* 2018; Zheng *et al.* 2018), and the short hypocotyls might not be directly derived from altered phyB signaling. This is also supported by the fact that viable quadruple *mlk*/*ppk* knockout mutants could not be generated yet, while the loss of *PHYB* is certainly not lethal.

SPA1 (SUPPRESSOR OF PHYA-105 1) possesses kinase activity and regulates PIF1 and 4 phosphorylation (Paik *et al.* 2019; Lee *et al.* 2020). However, currently, it is unclear if SPA proteins serve as kinases that directly transfer R light-induced phyB activity into PIFs phosphorylation to promote their degradation.

SOS2 (SALT OVERLY SENSITIVE 2) is a serine/threonine kinase that phosphorylates and activates SOS1 under salt stress. Since SOS1 is a plasma membrane–localized Na^+/H^+ antiporter, its activation leads to a decrease of the cytosolic Na^+ concentration (Mahajan *et al.* 2008). Additionally, it was recently demonstrated that SOS2 is also active within the nucleus, where it phosphorylates PIF1 and 3. Both transcription factors foster salt sensitivity. SOS2 interacts with the 2 PIF proteins and phyB in nuclear bodies, indicating that they altogether colocalize in photobodies. Phytochromes are able to activate SOS2 to induce PIF1 and 3 phosphorylation and reduce their stability to increase salt tolerance (Ma *et al.* 2023). Notably, there are also reports that describe negative effects of phyB on salt stress tolerance (Kwon *et al.* 2018; Yang *et al.* 2018; Liu *et al.* 2023).

In another study, it was shown that SOS2 also phosphorylates PIF4 and 5 (Han *et al.* 2023). Surprisingly, this does not lead to increased PIF turnover, as in the case of PIF1 and 3, but to an increased stability of PIF4 and 5. By this means, SOS2 can act as a positive regulator of hypocotyl elongation in low R:FR and counter the reduced growth response during

salt stress (Hayes *et al*. 2019; Han *et al*. 2023). SOS2 interacts with PIF4 and PIF5 within nuclear bodies, but at this point it is unclear if these are formed by phyB.

The mitogen activated protein kinase MPK6 (MAP KINASE 6) and PIF3 antagonistically regulate cotyledon opening. MPK6 can phosphorylate PIF3 in vitro and is activated by R light through phosphorylation by MKK10 (MAP KINASE KINASE 10). MPK6 and PIF3 are colocalized in nuclear bodies (Xin *et al*. 2018). Nevertheless, it is not shown yet if these speckles have phyB photobody identity. Additionally, we currently do not understand how MKK10, MPK6, and phyB work together to mediate PIF3 phosphorylation.

Besides the herein described kinases, PIFs were also shown to be phosphorylated by BIN2 (BRASSINOSTEROID-INSENSITIVE 2) (Bernardo-García *et al*. 2014; Ling *et al*. 2017) and CK2 (CASEIN KINASE 2) (Bu *et al*. 2011), but recruitment to photobodies has not been studied yet. Altogether, it is likely that PIFs are phosphorylated by a concert of kinases; some act downstream of phyB in photobodies, while others likely function to integrate PIF activity into connected signaling pathways.

PIF phosphorylation is reversible, but the underlying regulatory mechanisms are very little understood. PP6 phosphatases and TOPP4 (TYPE ONE SERINE/THREONINE PROTEIN PHOSPHATASE 4) were reported to interact with diverse PIFs and regulate their phospho-status and protein stability (Yue *et al*. 2016; Yu *et al*. 2019). Currently, it is unknown if PIFs get dephosphorylated within photobodies or after their release from photobodies. Both mechanisms could coexist, as indicated by bimolecular fluorescence complementation; in the dark, TOPP4 binds to PIF3 in the nucleoplasm, while R light relocates their interaction to nuclear speckles (Yue *et al*. 2016). However, sequestering phosphatases into photobodies could also represent a potential mechanism to inactivate these enzymes and limit PIF activity.

Recruitment of E3 ubiquitin ligases by photobodies

A plethora of E3 ubiquitin ligases initiate proteasomal degradation of proteins by polyubiquitination. Hereby, the substrate recognition by the E3 provides specificity to the degradation process (Mazzucotelli *et al*. 2006). There are several E3 ligase candidates that could induce PIF degradation within photobodies after phyB activation.

The LRB 1 to 3 (LIGHT-RESPONSE BTB1 to 3) form a small family of BTB (Broad-complex, Tramtrack, and Bric-à-brac) proteins that serve as substrate recognition adaptors of CULLIN3-based E3 complexes. The family was initially identified in a reverse genetic screen and described as negative regulators of phyB protein abundance and hence R light signaling (Christians *et al*. 2012). However, a subsequent study revealed that LRBs not only promote phyB turnover but also initiate PIF3 degradation after its phosphorylation in response to R light (Ni *et al*. 2014). In the dark and in R light, LRB1 and 2 are evenly distributed within the nucleoplasm and do not seem to be recruited to photobodies (Christians *et al*. 2012). This suggests that phyB activity leads to the recruitment of other E3 s into photobodies or that PIF3 is released from photobodies after phosphorylation to initiate its degradation via LRB-mediated polyubiquitination within the nucleoplasm. The second option is supported by the observation that mimicking phosphorylation of PIF3 increases its turnover even in etiolated seedlings (Ni *et al*. 2013), where photobodies are absent. Nevertheless, both alternatives are certainly not mutually exclusive, especially since in recent years several other E3 ligases were identified that target PIF proteins. For instance, phyB utilizes EBF1 and 2 (EIN3-BINDING F BOX PROTEIN 1 and 2) to induce degradation of PIF3 and of EIN3 (ETHYLENE-INSENSITIVE 3) to promote photomorphogenesis via 2 distinct molecular mechanisms: The phyB-dependent phosphorylation of PIF3 is sufficient to induce the recognition by EBF1/2 without further requirement of phyB activity (Dong *et al*. 2017). In contrast, phyB seems to directly enhance the binding affinity of EBF1/2 to EIN3 by acting as a scaffold for the transcription factor and the F-box proteins (Shi *et al*. 2016).

We currently do not know if LRBs and EBFs are recruited to photobodies. However, other E3 ligases that control PIFs were at least shown to be located in some type of nuclear speckles. PIF1 interacts with the F-box protein CTG10 (COLD TEMPERATURE GERMINATING 10) and PIF4 binds to BOP2 (BLADE-ON-PETIOLE 2) within nuclear localized speckles in bimolecular fluorescence complementation assays. CTG10 negatively regulates PIF1 levels to induce germination and promote photomorphogenesis, while BOP1 and 2 reduce PIF4 abundance to lower the response to higher ambient temperatures and promote photomorphogenesis (Zhang *et al*. 2017; Majee *et al*. 2018).

The RING finger E3 ubiquitin ligase HOS1 (HIGH EXPRESSION OF OSMOTICALLY RESPONSIVE GENE 1) is mainly located at the nuclear envelope, which is in agreement with its function in nuclear-cytoplasmic mRNA export (Tamura *et al*. 2010; Lazaro *et al*. 2012; MacGregor *et al*. 2013). However, interaction with at least 1 of its protein substrates is concentrated in nuclear bodies: HOS1 promotes turnover of the flowering regulator CO (CONSTANS), which seems to be mediated by phyB (Jung *et al*. 2012; Lazaro *et al*. 2012, 2015). Interestingly, HOS1 also represses PIF4, however, without inducing its turnover. Instead, HOS1 is phyB dependently recruited to PIF4 DNA binding sites and inhibits PIF4's transactivation activity (Kim *et al*. 2017).

The best characterized E3 ligases that regulate light responses are COP1-SPA complexes. The *Arabidopsis* genome encodes for 1 *COP1* (*CONSTITUTIVE PHOTOMORPHOGENIC 1*) gene and for 4 *SPA* paralogues. COP1-SPA complexes form E3 ligases that initiate the degradation of positive regulators of photomorphogenesis. Many COP1-SPA substrates are transcription factors, like HY5, HYH, PIL1, and HFR1 (Osterlund *et al*. 2000; Holm *et al*. 2002; Duek *et al*. 2004;

Luo et al. 2014). In addition, COP1-SPA complexes functionally interact with DET1 (DE-ETIOLATED 1) (Nixdorf and Hoecker 2010). *Arabidopsis* plants with a loss of *DET1* function were the very first described mutants that showed a de-etiolated/constitutive photomorphogenic phenotype in darkness (Chory et al. 1989). Like COP1 and SPAs, DET1 forms CULLIN4-based E3 complexes that are necessary to suppress photomorphogenesis in the dark (Lau and Deng 2012). Currently, DET1 is functionally less understood than COP1 and SPA proteins. However, a recent study suggested that DET1 and COP1 jointly work in HY5 degradation, since DET1 is able to interact with both COP1 and HY5 and fosters the interaction between HY5 and COP1 and, hence, HY5 degradation (Cañibano et al. 2021).

In addition to their role in degradation of positive light signaling components, COP1, DET1, and SPAs stabilize PIF transcription factors (Bauer et al. 2004; Leivar et al. 2008b; Dong et al. 2014; Shi et al. 2015; Gangappa and Kumar 2017; Pham et al. 2018). For example, COP1 and SPAs prevent PIF3 phosphorylation by BIN2 kinases that otherwise mark PIF3 for degradation (Ling et al. 2017). Altogether, COP1, DET1, and SPAs act PIF dependently and independently to promote skotomorphogenesis, thermomorphogenesis, and shade avoidance and hence act as negative regulators of phyB activity.

Different light receptors inactivate COP1-SPA complexes by at least 3 mechanisms: (1) nuclear exclusion of COP1, (2) degradation of SPA subunits, and (3) light receptor–SPA protein interaction to prevent COP1-SPA complex formation. COP1 nuclear exclusion by R light was described in the past but is disputed by more recent reports (Osterlund and Deng 1998; Jang et al. 2010; Balcerowicz et al. 2017). Nevertheless, COP1 re-accumulates in the nucleus under canopy shade (Pacín et al. 2013). In addition, phyB can sequester SPA proteins to prevent COP1-SPA binding and initiate the degradation of SPAs (Lu et al. 2015; Sheerin et al. 2015). COP1 and SPA proteins colocalize in nuclear speckles that can have phyB photobody identity (Seo et al. 2003; Zheng et al. 2013; Kim et al. 2023a). This indicates that in analogy to PIF inactivation, photobodies serve to sequester, inactivate, and degrade these negative regulators of photomorphogenesis. In addition, photobodies seem to functionally repurpose COP1 and SPAs by turning them into positive regulators of phyB activity: COP1 is required for the R light-induced degradation of SPA2 and potentially other SPAs (Chen et al. 2015). Additionally, COP1 and SPA proteins are involved in the phosphorylation and degradation of PIFs in response to R light (Zhu et al. 2015; Pham et al. 2018; Paik et al. 2019). However, based on *cop1*'s de-etiolated phenotype, its role in suppressing phyB action is more profound than its role in promoting R light signaling. In agreement, COP1 is also a negative regulator of photobody formation and maintenance by regulating structural components of these subnuclear domains.

Structural photobody components regulate gene expression

As stated above, in the dark, the Pfr form of phyB slowly switches back into its inactive Pr form, a process called dark or thermal reversion. Interestingly, the in vivo dark reversion is much slower than the reversion in vitro (Rausenberger et al. 2010). This indicates that phyB modifications or phyB binding proteins decelerate the dark reversion rate. One of these phyB interactors is PCH1 (PHOTOPERIODIC CONTROL OF HYPOCOTYL 1), a positive regulator of R light-mediated photomorphogenesis (Huang et al. 2016b). PCH1 not only directly interacts with phyB and is part of photobodies but also stimulates the formation of large photobodies and the maintenance of photobodies during the night. Hence, overexpression of *PCH1* accelerates the appearance of photobodies after activating phyB and prevents photobody dissociation in the dark. It turned out that PCH1 and its paralogue PCHL (PCH1-LIKE) slow down the dark/thermal reversion of phyB by stabilizing phyB's Pfr form without interfering with its intrinsic R-FR photoconversion (Fig. 4) (Enderle et al. 2017; Huang et al. 2019). Stabilizing the Pfr form and hence photobodies extends phyB activity and therefore R light signaling in the night. Therefore, *pch1* mutants show long hypocotyls in short-day conditions and in long days that coincide with elevated temperatures (when the dark/thermal reversion is accelerated). Since the Pfr form of phyB sequesters PIFs and initiates their degradation, PIF4 protein levels in *pch1* are increased during the night (Huang et al. 2019). This could also be the consequence of increased *PIF4* transcript levels in *pch1*. Altogether, the increased PIF4 levels go along with higher PIF target gene expression. Furthermore, PCH1 and PCHL seem to regulate PIF activity also by another mechanism, since they not only regulate hypocotyl growth in the night but also various R light responses, including seed germination and chlorophyll biosynthesis (Cheng et al. 2020). Interestingly, PCH1/PCHL negative influence on PIF1 abundance is only partial phyB dependent: PCH1/PCHL directly interact with PIF1 in a phyB-independent way. Additionally, in *PCH1/PCHL* overexpressors, PIF1 protein levels are lower even in etiolated seedlings before any light inputs and hence prior phytochrome activity. This phyB-independent function has consequences for PIF1 DNA binding and target gene expression: both are reduced in imbibed seeds of *PCH1* overexpressors after phyB inactivation by FR light. Nevertheless, phyB binds stronger to PIF1 in the presence of PCH1/PCHL. The latter might be due to a bridging activity by PCH1/PCHL or due to the acceleration of photobody formation by these proteins.

In addition to phyB and PIFs, PCH1 also interacts with members of the TPL/TPR (TOPLESS/TOPLESS-RELATED) family, a group of transcriptional corepressors. These proteins are recruited to target genes by various transcription factors to negatively regulate gene expression (Plant et al. 2021). phyB alone does not bind TPL protein, and hence PCH1 serves as a primary photobody client to pull TPL and

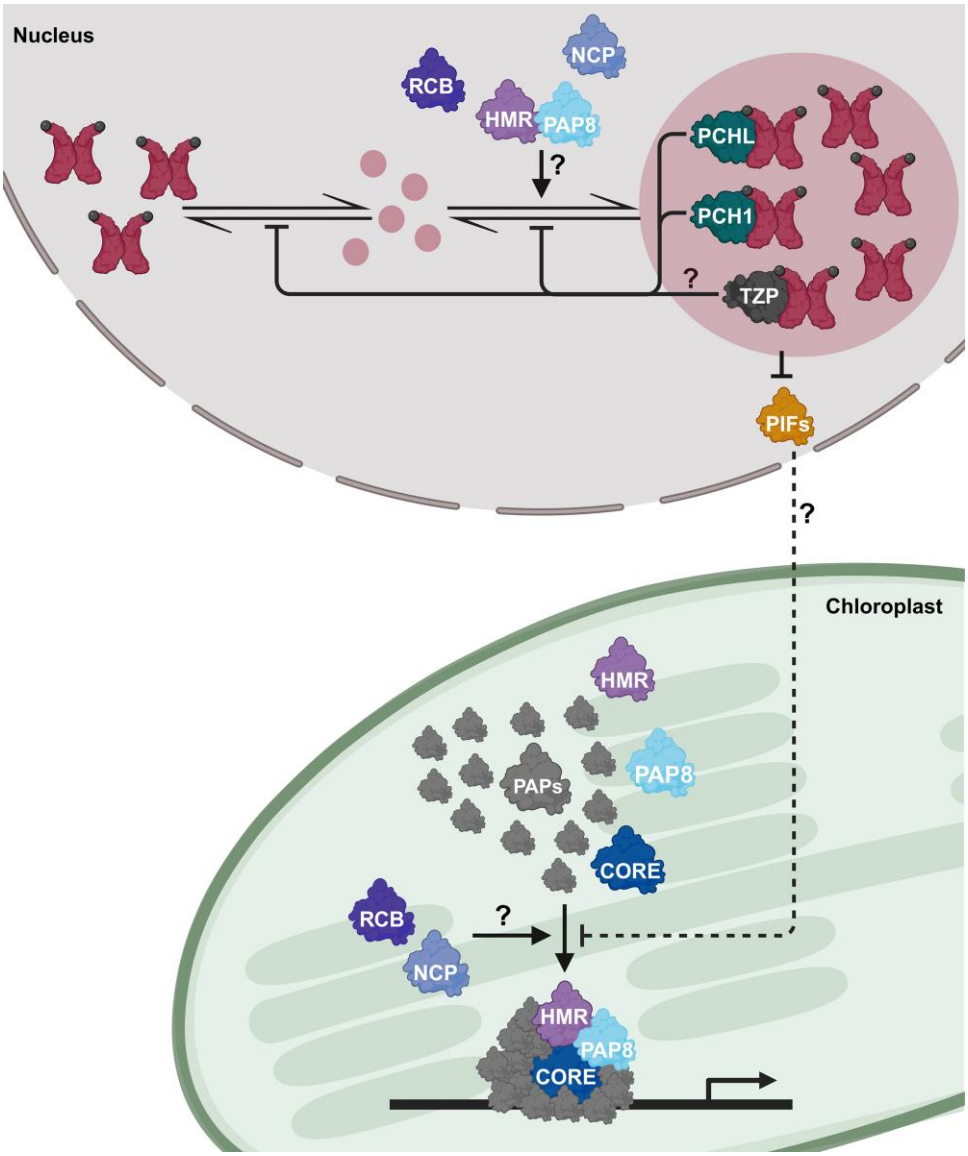

Figure 4. Function of photobody regulators. PCH1 and PCHL stabilize phyB's Pfr form and hence prevent photobody disassembly. TZP also regulates photobody formation and growth, but it is currently unknown if this happens through the stabilization of phyB's Pfr form as well. HMR, PAP8, RCB, and NCP are located within the nucleoplasm and chloroplasts. They exhibit a dual function, since they promote photobody growth and the assembly of plastid-encoded RNA polymerase complexes consisting of core proteins and PAPs. Additionally, the formation of the PEP holoenzyme complex is regulated by PIF-dependent nucleus-to-plastid signaling.

related proteins into photobodies. Interestingly, multiple *tpl*/*tpr* mutants show reduced hypocotyl growth in R light, indicating that TPL/TPR-dependent gene repression serves to decrease phyB signaling (Kim *et al.* 2023a). Hence, incorporation of TPL/TPR proteins into photobodies could be a means of preventing their interaction with transcription factors or could serve to isolate transcription factor-TPL/TPR complexes from their DNA binding sites. PCH1/PCHL also interact with COP1, which can lead to COP1-induced PCH1/PCHL ubiquitination and subsequent degradation in the dark (Cheng *et al.* 2020). In consequence, COP1 could promote the dark/thermal reversion of phyB by lowering PCH1/PCHL levels and thus decreasing Pfr stability.

Through binding of PIFs, COP1-SPA complexes and TPL/TPR proteins, photobodies regulate transcription indirectly. However, photobodies themselves are locations of active gene expression as work on TZP (TANDEM ZINC KNUCKLE PROTEIN) has demonstrated. TZP was originally identified in a study of natural variations for hypocotyl elongation and encodes a protein with 2 types of nucleotide binding domains: 1 PLUS3 and 2 zinc finger domains (Loudet *et al.* 2008). TZP is recruited by phyB into photobodies which are disrupted by chemicals that are known to block transcription (Kaiserli *et al.* 2015). This is an indication that photobodies are associated with transcriptional processes. Furthermore, TZP binds single, but not double stranded DNA in vitro,

which might indicate that TZP binds to separated DNA strands in active chromatin regions. This is supported by TZP's binding to 2 loci of floral activators (*CO* and *FT*). TZP's DNA binding and TZP-dependent gene activation is inhibited in the absence of photobodies (by FR light treatment or loss of phyB function), supporting the hypothesis that TZP-dependent gene regulation is happening in transcriptionally active photobodies (Kaiserli *et al*. 2015).

As described above for PCH1/PCHL, TZP also physically interacts with COP1, which facilitates TZP degradation (Li *et al*. 2022a). However, this is not the only common feature of PCH1/PCHL and TZP, since *tzp pch1* double mutants synergistically suppress R light signaling in the context of hypocotyl growth (Fang *et al*. 2022). In *tzp pch1* double mutants, PIF4 levels do not only accumulate during the dark phase but also during the day, even though *PIF4* transcription is barely affected in these mutants. In consequence PIF target gene expression is upregulated in *tzp pch1*. TZP nuclear body formation is impaired in *pch1*, which is not surprising, since TZP requires phyB to be incorporated into photobodies and PCH1 promotes phyB photobody formation. However, TZP is a structural component of photobodies as well, since photobody formation and growth are also negatively affected in *tzp* mutants (Fang *et al*. 2022), but it is not demonstrated yet if this also happens through the stabilization of phyB's Pfr form (Fig. 4).

The dual function of photobody regulators

After the discovery of phyB photobodies, forward genetic screens led to the identification of regulators of photobody formation. The first protein that was reported to be one of these regulators was HMR (HEMERA), a nucleus and plastid dual-localized protein (Chen *et al*. 2010). HMR was identified in a confocal-based forward genetic screen in a mutagenized *PHYB:GFP* background. Loss of *HMR* does not affect phyB protein levels but affects the size of phyB photobodies, which have a much smaller size than photobodies in the wild-type under continuous red light (Chen *et al*. 2010). HMR directly interacts with phyB in a light-dependent manner (Galvão *et al*. 2012). In addition, HMR directly interacts with and regulates the degradation of PIF1 and PIF3 as a transcriptional coactivator (Qiu *et al*. 2015).

HMR is also known as PAP5/pTAC12 (PEP-ASSOCIATED PROTEIN 5/PLASTID TRANSCRIPTIONALLY ACTIVE 12), since it is one of the associated proteins of the plastid-encoded RNA polymerase (PEP) complex. HMR/PAP5/pTAC12 was co-purified with the plastid transcriptionally active chromosome (pTAC) fraction from chloroplasts (Pfalz *et al*. 2006; Pfannschmidt *et al*. 2015). This is why *hmr* mutants show a combination of tall hypocotyl and albino phenotypes due to defects in phyB signaling in the nucleus and in PEP activity that is essential for chloroplast biogenesis. Interestingly, it has been demonstrated that HMR is first targeted to plastids, where it is processed to its mature form and from there it is relocated to the nucleus (Nevarez *et al*. 2017). Altogether, these studies propose that once photoactivated phyB is translocated into the nucleus, HMR promotes the formation of large phyB photobodies in the nucleus while HMR is also assembled into the PEP complex in chloroplasts (Fig. 4).

The dual role of HMR in photobody formation in the nucleus and PEP activation in plastids is in line with the nuclear control of chloroplast biogenesis. A recent report showed that photo-activated phytochromes in the nucleus send a nucleus-to-plastid signal to trigger the assembly of PEP complexes and chloroplast biogenesis by promoting PIF degradation (Yoo *et al*. 2019). Accordingly, forward genetic screens for *hmr*-like tall-and-albino mutants defective in phytochrome-mediated control of the PEP assembly identified 2 novel phytochrome signaling components: REGULATOR OF CHLOROPLAST BIOGENESIS (RCB) and NUCLEAR CONTROL OF PEP ACTIVITY (NCP) (Yang *et al*. 2019; Yoo *et al*. 2019). Like *hmr* mutants, both *rcb* and *ncp* mutants are defective in the formation of large photobodies, leading to the accumulation of PIF1 and PIF3 (Fig. 4) (Yang *et al*. 2019; Yoo *et al*. 2019). RCB/NCP-dependent photobody formation and PIF degradation in the nucleus triggers the assembly and activation of the PEP complex in chloroplast biogenesis (Yang *et al*. 2019; Yoo *et al*. 2019). Although they are dual-localized proteins, RCB regulates the PEP assembly and complex primarily in the nucleus via nucleus-to-plastid signaling, while NCP located in plastids has an essential role in PEP assembly. Both proteins contain noncatalytic thioredoxin-like domains in their C terminus. The biochemical functions of RCB and NCP in regulating photobody formation and the assembly of the PEP complex are still unknown.

Recently, another subunit of the PEP complex, PAP8/pTAC6, was reported to affect photobody formation presumably by acting together with HMR/PAP5/pTAC12. Mutation of *PAP8* suppresses the short hypocotyl phenotype of *PHYB:GFP* overexpressors under red light and leads to defects in the formation of large photobodies (Liebers *et al*. 2020). PAP8 is also targeted to both the nucleus and chloroplasts, where it directly interacts with HMR/PAP5. Since the size of nuclear PAP8 is similar to the transit peptide-cleaved form of chloroplast PAP8, this study proposes that PAP8 is translocated from chloroplasts to the nucleus. Here, PAP8 physically interacts with HMR to promote the formation of large photobodies (Fig. 4). The regulation of photobody formation by PAP8 might work through HMR because the direct interaction between PAP8 and phyB has not been demonstrated yet.

It is worth pointing out that all these regulators of photobody stability share similar characteristics. First, HMR, RCB, NCP, and PAP8 are all dual-localized proteins in the nucleus and in chloroplasts. Second, they are required for the formation of large photobodies without affecting phyB protein level or nuclear localization. Third, they are essential for chloroplast biogenesis since all mutants are albinos. Fourth, HMR and PAP8 are essential components of the PEP

complex, while RCB and NCP are required for PEP assembly even though they are not thought to be components of the PEP complex. Fifth, none of these factors have been identified as structural components of photobodies. Altogether, these factors suggest that although the regulatory mechanism of photobody stability by these proteins is still unclear, the formation of large photobodies is tightly linked to the formation of the PEP holoenzyme complex via PIF-dependent nucleus-to-plastid signaling (Fig. 4) (Hwang *et al.* 2022). The nature of this anterograde signal is unknown; however, photobody stability during light exposure must be important to initiate light-triggered chloroplast biogenesis in a seedling's early development. It is also worth mentioning that these regulators are all plant-specific proteins evolved during land adaptation and do not exist in cyanobacteria or green algae (Chen *et al.* 2010; Nishiyama *et al.* 2018; Yang *et al.* 2019). Their regulatory mechanisms on photobody stability might have evolved to optimize photomorphogenesis including chloroplast biogenesis during terrestrial adaptation.

Photobodies recruit splicing factors to regulate gene expression

A cell's precursor mRNAs (pre-mRNAs) need to undergo splicing in order to form mature mRNAs that serve as templates for protein production. Hence, introns must be removed and exons must be fused. These reactions are mediated by the major spliceosome, which consists of over 100 proteins and several small protein–RNA complexes, called small nuclear ribonucleoproteins. These include U1, 2, 4, 5, and 6 that recognize splicing signals within pre-mRNA molecules (Will and Lührmann 2011; Matera *et al.* 2014; Lee and Rio 2015).

Mandatory retention of exons and removal of introns is called constitutive splicing, while the inclusion of introns, the exclusion of exons, or the use of nonconstitutive splice sites are called alternative splicing. Alternative splicing produces multiple transcripts from a single gene by using different splice sites. Splicing factors such as SR proteins and heterogenous nuclear riboproteins promote or prevent access to these splicing signals. Therefore, they give specificity to each splicing process and determine the produced mature mRNA isoform (Reddy *et al.* 2013; Lee and Rio 2015). Interestingly, this specificity is regulated by photobodies.

About 7% of all *Arabidopsis* genes undergo phytochrome A and B-dependent alternative splicing after R light exposure for up to 3 h (Shikata *et al.* 2014). For example, phytochromes promote alternative splicing of *SPA3* transcripts. This leads to a loss of SPA3's C terminus due to the formation of premature termination codons. Unlike its full-length isoform, the truncated SPA3 proteins act as repressors of hypocotyl elongation (Shikata *et al.* 2014). Another example of phyB-mediated alternative splicing affects PIF3 translation. The 5′UTR of *PIF3* contains 2 introns. Within the second intro lies an upstream open reading frame. R light and phyB promote retention of this upstream open reading frame in the spliced *PIF3* mRNA, which negatively affects PIF3 translation (Dong *et al.* 2020).

These 2 examples demonstrate how phytochrome-activity promotes R light signaling through alternative splicing. So far, 6 phyB interacting splicing factors were identified. For 2 heterogenous nuclear riboproteins that regulate alternative splicing in the moss *Physcomitrella patens* (Shih *et al.* 2019; Lin *et al.* 2020), no photobody localization has been assessed yet. However, 4 different splicing factors were identified in *Arabidopsis*. They colocalize with phyB in nuclear speckles, indicating that photobodies directly influence pre-mRNA splicing.

RRC1 (*REDUCED RED-LIGHT RESPONSES IN CRY1CRY2 BACKGROUND 1*) was identified in a screen for R light hyposensitive mutants (Shikata *et al.* 2012). *RRC1* encodes for an ortholog of the human splicing factor SR140. RRC1 and SR140 are SR proteins that carry a serine and arginine-rich domain at their C termini (RS domain) and an N-terminal RNA recognition domain (RRM domain). Loss of RRC1's C-terminal RS domain leads to reduced phyB signaling. These mutations or loss of *PHYB* affect alternative splicing of several SR protein genes in response to R light. However, *rrc1* null mutants show pleiotropic developmental defects, which indicates that RRC1's function is not limited to phyB signaling (Shikata *et al.* 2012).

RRC1 interacts with SFPS (SPLICING FACTOR FOR PHYTOCHROME SIGNALING). Both splicing factors have several features in common (Xin *et al.* 2017, 2019). Like *RRC1*, *SFPS* was also identified in a screen for R light hyposensitive mutants. SFPS is related to the human splicing factor SPF45. The human orthologues of RRC1 and SFPS (SR140 and SPF45) associate with the small nuclear ribonucleoprotein U2 and the same seems to be true for RRC1 and SFPS (Xin *et al.* 2017, 2019). U2 and its associated factors bind to 3′ splice sites and intronic branch points (typically located up to 50 bp upstream of the 3′ splice site). Hence, pre-mRNA binding by U2 and its associated factors define the actual 3′ splice site of each individual splicing reaction (Wahl *et al.* 2009; Matera *et al.* 2014). Interestingly, the interaction of RRC1 and SFPS is conserved, since human SPF45 and SR140 were later found to interact as well (Martín *et al.* 2021), which supports the notion that these splicing factors act jointly in various signaling pathways.

The above-mentioned similarities between RRC1 and SFPS go even further. Both proteins were shown to interact with phyB and to locate in nuclear speckles that at least partly colocalize with phyB photobodies (Xin *et al.* 2017, 2019). However, in the case of RRC1 colocalization and interaction with phyB could not be confirmed in all studies (Shikata *et al.* 2012; Kim *et al.* 2023b), which could be explained by differing experimental conditions.

In *rrc1* and *sfps* mutants, splicing of genes that are associated with light stimulus, photosynthesis, and the circadian clock is impaired. One example is that *ELF3*: *rrc1* and *sfps* mutants contain higher levels of not fully spliced *ELF3* transcripts that encode for a nonfunctional ELF3 protein (Xin *et al.* 2017,

2019; Kim et al. 2023b). The alternative splicing of clock gene transcripts in *rrc1* leads to misregulation of rhythmic gene expression and in consequence to higher PIF4, PIF5, and PIF7 protein levels and increased hypocotyl growth (Kim et al. 2023b).

Both RRC1 and SFPS bind independently and jointly to the splicing factor SWAP1 (SUPPRESSOR-OF-WHITE-APRICOT/SURP RNA-BINDING DOMAIN-CONTAINING PROTEIN1) (Kathare et al. 2022). As shown for RRC1 and SFPS, SWAP1 also interacts with phyB and colocalizes with phyB and U2 subunits in nuclear speckles. Altogether, phyB might recruit RRC1, SFPS, and SWAP1 to photobodies to concentrate these splicing factors to induce splicing of light signaling and circadian clock genes. The increased concentration of particular splicing factors could enhance the efficiency of specific splicing reactions to promote R light signaling. The association of RRC1, SFPS, and SWAP1 with U2 suggests that they link phyB activity with the selection of 3′ splice sites. It is worth pointing out that an alternative model was proposed in which phyB and RRC1 promote light responses spatially separated. While *PHYB* expression within the epidermis is sufficient to repress hypocotyl growth, *RRC1* seems to act mainly within the endodermis to promote R light responses (Kim et al. 2023b).

While RRC1, SFPS, and SWAP1 act as positive components of phyB signaling, SMP2 (SWELLMAP 2) is a splicing factor that suppresses photomorphogenesis—it antagonizes phyB signaling to promote hypocotyl elongation (Yan et al. 2022). SMP2 was found as a phyB interactor in a yeast two-hybrid screen using the C-terminal output module. It is a homolog of human Slu7 that is also involved in 3′ splice site selection. SMP2 was found to regulate *RVE8* (*REVEILLE 8*) pre-mRNA splicing. RVE8 is a transcription factor that induces the expression of several transcription regulators involved in hypocotyl growth repression (Hsu et al. 2013). Alternative splicing of *RVE8* pre-mRNA can lead to non-functional mature mRNA isoforms (James et al. 2012). In *smp2*, more functional *RVE8* mRNA isoforms are produced, which might explain the reduced hypocotyl growth in these mutants. SMP2 is localized in the nucleoplasm and in nuclear speckles, and some of these speckles exhibit photobody identity, since they colocalize with phyB (Yan et al. 2022). In analogy to the sequestration of other negative regulators of phyB signaling (like PIFs or SPAs), photobodies might isolate SMP2 from cofactors or pre-mRNA substrates to impair its activity.

Regulation of chromatin dynamics by photobodies

Active phyB also regulates gene expression by modifying gene activity on a macromolecular level. For example, phyB is involved in light-dependent chromatin compaction. Here, phyB activity stimulates the formation of highly condensed heterochromatin domains (chromocenters) that are transcriptionally inactive (Tessadori et al. 2009; van Zanten et al. 2010). In addition to chromatin compaction, phyB modifies the location of light-induced genes within the nucleus as shown for the reposition of the *CAB* (*CHLOROPHYLL A/B BINDING PROTEIN*) gene cluster. This gene cluster moves phyB dependently from the nuclear interior to the nuclear periphery. Since this repositioning seems to happen before the gene cluster is fully transcriptionally induced, it might be a mechanism to increase light-dependent gene expression. In contrast, in the dark, PIFs retain these light-inducible loci in the nuclear interior (Feng et al. 2014). This suggests that phyB activation and photobody formation induce PIF degradation to translocate light-inducible loci. It is possible that these loci move into the proximity of nuclear pore complexes to promote transcription, since these structures are not only important for gene activation in yeast and metazoan systems, but also in plants (Tamura 2020). However, it needs to be tested, if phyB-dependent gene reposition colocalizes with photobodies and/or nuclear pore complexes.

Another antagonistic function of phyB and PIFs is the control of nucleosome occupancy by the histone variant H2A.Z. Nucleosomes consist of a histone octamer and approximately 146 base pairs of DNA that are wrapped around it (Luger et al. 1997). The octamer is formed by 4 different histones, including H2A. H2A can be exchanged by H2A.Z within gene bodies, and by this means, gene expression and gene responsiveness is regulated. In *Arabidopsis*, higher H2A.Z levels within gene bodies are found to correlate with reduced gene activity (Coleman-Derr and Zilberman 2012; Yelagandula et al. 2014; Sura et al. 2017; Carter et al. 2018). Two evolutionary conserved chromatin remodeling complexes can exchange H2A for H2A.Z and vice versa. The SWR1 complex incorporates H2A.Z into nucleosomes, while the INO80 complex evicts H2A.Z and deposits H2A (Mizuguchi et al. 2004; Papamichos-Chronakis et al. 2011). These H2A-H2A.Z dynamics enable transcriptional responses to various environmental stimuli in plants, including light. Mutations of *INO80* cause R light hypersensitivity, in part due to reduced H2A.Z occupancy at the *HY5* gene body, which leads to increased expression of *HY5* (Yang et al. 2020). However, SWR1 and INO80 complexes need to be recruited to light-responsive genes. Several recent studies have identified antagonistic phyB and PIF-dependent mechanisms to regulate H2A.Z occupancy. phyB was found to be able to bind 2 subunits of the SWR1 complex (SWC6 and ARP6) in a R light-dependent fashion (Wei et al. 2021). Using in vitro assays, the photosensory module and the output module interacted independently with both subunits. However, in planta interactions were found only in the presence of R light, indicating that the photobody forming Pfr form is necessary for these interactions. Like loss of *PHYB* function, mutations of both SWR1 subunits show light-hyposensitive growth and increased expression of auxin biosynthesis and response genes, suggesting that the SWR1 complex represses growth by lowering auxin biosynthesis. This is supported by the R light and SWR1-dependent deposition of H2A.Z at the *YUCCA9* locus.

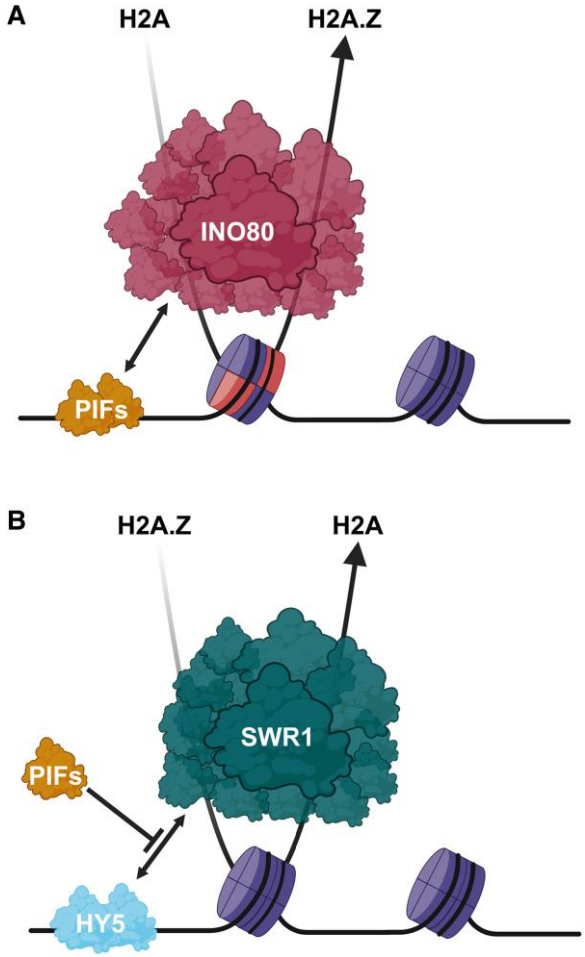

Figure 5. Negative regulation of H2A.Z occupancy by PHYTOCHROME-INTERACTING FACTORs. **A)** PIF4 and 7 recruit the INO80 complex to promote H2A.Z eviction and gene activity of PIF target genes in response to low R:FR or elevated temperatures. PIF4, 5, and 7 can interact with the small INO80 complex subunit EEN (EIN6 ENHANCER), a homolog of Ies6 (Ino eighty subunit 6) found in yeast. PIF4 was also shown to interact with the N-terminal region of INO80 itself. **B)** HY5 interacts with 2 SWR1 complex subunits, ARP6 and SWC6, to recruit the chromatin remodeling complex. This promotes H2A.Z incorporation into nucleosomes. Several PIFs physically interact with SWC6 as well, which seems to compete with the HY5's SWR1 recruitment.

Interestingly, this incorporation of H2A.Z is not only phyB but also HY5 dependent (Wei *et al.* 2021). The cause for the HY5 dependency is likely HY5's ability to bind to SWC6 and ARP6 as well. This interaction allows the recruitment of SWR1 complexes to HY5 target loci (Fig. 5) (Mao *et al.* 2021). Altogether, this indicates that light-dependent H2A-H2A.Z dynamics happen upstream (Yang *et al.* 2020) and downstream (Mao *et al.* 2021; Wei *et al.* 2021) of *HY5*.

PIF7 is the major PIF for the induction of hypocotyl growth in response to low R:FR (Li *et al.* 2012; Willige *et al.* 2021). After exposure to low R:FR, PIF7 gets dephosphorylated and changes its subnuclear localization. While PIF7 is concentrated in large photobodies in white light, it is released from these domains after low R:FR exposure (Willige *et al.* 2021; Xie *et al.* 2023). These responses likely precede PIF7 DNA binding to its target genes. These target genes are highly enriched in transcriptional regulators, suggesting that PIF7 acts upstream of a transcription factor cascade. Interestingly, 5 minutes of low R:FR exposure is sufficient to induce PIF7 DNA binding, while longer exposure times intensify this binding and lead to more binding events (Willige *et al.* 2021). This rapid DNA binding is accompanied by H2A.Z removal from gene bodies of PIF7 target genes. Hereby, PIF7 DNA binding and H2A.Z seem to precede the low R:FR-induced transcriptional activation of these genes. Importantly, this H2A.Z eviction in response to low R:FR is strongly compromised in *pif457* mutants, demonstrating its PIF dependency.

EEN (EIN6 ENHANCER), the ortholog of the yeast and human Ies6 (INO80 Subunit 6) protein, is a small but essential subunit of the INO80 complex (Zander *et al.* 2019). Based on available cryo-EM structures of the INO80 complex, Ies6 is directly involved in nucleosome binding by the INO80 complex (Watanabe *et al.* 2015; Ayala *et al.* 2018; Eustermann *et al.* 2018). At least PIF4, 5, and 7 can interact with EEN, and *een* mutants show hyposensitivity to low R:FR. Additionally, *een* exhibits defects in low R:FR induced H2A.Z eviction (Willige *et al.* 2021). This indicates that PIFs recruit the INO80 complex via interaction with EEN to regulate H2A.Z removal and hence gene expression (Fig. 5). However, PIFs seem not only bind to EEN, since at least PIF4 also interacts with the N-terminal region of INO80, the largest subunit of the INO80 complex. In response to warm temperatures, these interactions mediate H2A.Z eviction at PIF4 target genes to facilitate gene activity and hence thermomorphogenesis (Xue *et al.* 2021).

Promoting H2A.Z eviction is not the only way PIFs regulate H2A.Z occupancy. It was shown that several PIFs are able to physically interact with SWC6, the aforementioned subunit of the SWR1 complex. This interaction happens within nuclear speckles, but at this point it is not clear if these foci are photobodies. Interestingly, PIF-SWC6 binding seems to compete with the HY5-SWC6 interaction that serves to recruit the SWR1 complex to HY5 target loci (Chen *et al.* 2023). Hence, besides facilitating H2A.Z eviction, PIFs also seem to prevent H2A.Z deposition that is promoted by HY5 (Fig. 5).

Overall, PIFs play an opposing role to phyB and HY5 in regulating H2A.Z occupancy. The promoters of photomorphogenesis interact with the SWR1 complex to deposit H2A.Z to reduce gene expression. In contrast, environmental signals such as low R:FR and warm temperatures reduce photobody formation/maintenance. In consequence, HY5 levels are reduced, but PIF activity is promoted. This leads to reduced H2A.Z occupancy and hence increased expression of PIF target genes.

Conclusions and perspectives

As summarized above, the current state of knowledge indicates that phyB photobodies are biochemical hotspots that

concentrate factors to accelerate enzymatic reactions and to give these reactions specificity. Conversely, this does not necessarily mean that these biochemical processes are inactive in the absence of photobodies since muted phyB responses can be observed even without photobody formation. Nevertheless, these nuclear foci are required to adjust the growth and development of plants to environmental conditions. In this context it is noteworthy that photobodies respond not only to light and temperature changes that directly influence the Pr-Pfr conversion. For example, photobodies are also influenced by salinity and jasmonate. Relatively little is understood about how these other abiotic and potentially biotic factors influence photobodies, if they modify the stability of phyB's Pfr form, or if they utilize other mechanisms to influence photobodies. In addition, it is unclear how all these signals are integrated to regulate the outcome of phyB signaling.

One key factor to unravel photobody signal integration is the understanding of the functional and compositional complexity of differing photobodies. One open question is if multiple speckles within a single nucleus are functionally similar and have overlapping protein compositions. These foci might interact with different genomic sites and fusion of photobodies could lead to intrachromosomal but also interchromosomal interactions. By this means, regulatory DNA elements could be brought into proximity of distant protein coding regions to allow flexible gene regulation. In contrast, multiple photobodies within a nucleus could be functionally specialized and might focus on certain functions of phyB signaling.

The finding that multiple speckles within a nucleus seem to have differing thermal stabilities indicates that they are compositionally diverse. The recently published mass spectrometry analysis of photobodies is a huge step in our understanding photobody composition. However, there are still many unknowns regarding not only photobody protein composition but also DNA and RNA sequences that might be structural components and determine photobody stability and size. Furthermore, we currently do not know if tissue- and cell type–specific photobodies exist that might provide another level of phyB signaling complexity.

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Author contributions

All authors contributed to writing and figure preparation.

Conflict of interest statement. None declared.

Data availability

There are no new data associated with this article.

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